ease, or whether, as in the case of the bacillus of hog cholera, the scientific world had not been considering the secondary organism as primary and overlooking some minute organism which might prove to be the real cause. Instead of using pure cultures, they began with feces from a typhoid patient, with the result that the young chimpanzee used developed a satisfactory attack of typhoid fever. Further, the fecal typhoid material was filtered, the filtrate given by the intestinal tract, and the rest injected under the skin. This was repeatedly unsuccessful. Not only was it impossible to infect in this way, but it was impossible to protect. Fifteen experiments out of sixteen, using pure cultures of human typhoid or mixed cultures of human and simian typhoid, were successful. The blood of the infected animals in every case developed rapidly agglutinative powers. Experimental typhoid in chimpanzees bears to the ordinary typhoid fever of adults much the same relation as does that of children. There is less prostration, less weakness, less anorexia and apparently no rose spots. The older the ape the more his disease resembles that with which we are familiar. In the three fatal cases that occurred in the series, autopsy showed marked enterocolitis, with great swelling of the Peyer's patches, but never ulceration. Numerous attempts with the infected material of the higher apes proved negative in the production of the disease in monkeys and in other laboratory animals. Metchnikoff and Besredka conclude that experimental typhoid in animals can be produced only in the higher apes and only by way of the intestinal tract. Their positive results were obtained by pure culture of the bacillus of Eberth, and they conclude that there exists no soluble virus to play a subsidiary or primary role in the infection. Vaccination which has proved sufficient to protect laboratory animals against peritoneal infection by Bacillus typhosus is powerless to prevent the typhoid fever of apes, which appears to be the real disease with which we are familiar.

Bone Lesions in Infective Diseases.—JOSEF KOCH (*Zeit. f. Hyg. u. Infekt.*, Band lxix, Heft, 3, 1911), who has been a strong advocate of the infective nature of rickets, brings forward further experimental work in support of that contention. In various kinds of experimental animals he has examined the localization of the injected bacteria in the bony structures. In 20 anthrax-infected animals great, sometimes enormous, masses of bacilli were found in the epiphyseal marrow, especially in the primary medullary spaces of the line of ossification, notably in close association with the vascular loops. Pneumococci appeared to frequent, in addition, the areas of endochondral ossification, and streptococci the perichondrium and periosteum. The great destruction of bacteria that goes on in the medulla is evidenced by the pigment seen in the endothelial cells, and the bacteria found at death constitute only a fraction of the bacteria that have existed there during the course of the infection. The histological changes which are evident as a result of the infection are very marked, both in compact and spongy bone. The spongy bone may be destroyed and the absorption of compact bone greatly hastened. In the general infections, although the heart blood may prove sterile, the juice of the epiphyseal medulla is by no means so, and frequently shows the presence of bacteria. Even in the acute infections of children, such as measles and whooping cough, the same thing is noted, thanks partly to the osteochondral boundaries in children being a site of predilection for infection. It is true that the pathogenic bacteria, after a time of increase, may be destroyed by specific antibodies, but this short time may be long enough to institute changes in the epiphyses; and the histological changes seen in rachitic bones are oftentimes of a regenerative kind. In 2 cases of florid rickets, with a fatal outcome, Koch found in the femoral epiphyses large numbers of bacteria, and he thinks that the cause of rickets is not to be sought in indefinite *noxæ*, but in bacteria.

Co-incident Abnormality of Numerous Organs of Internal Secretion.—F. HARBITZ (*Zentralbl. f. Allg. Path. u. Path. Anat.*, Band xxii, No. 18, September 30, 1911) reports a case that is very suggestive in its bearing upon the relation that exists among the glands of internal secretion. Autopsy was performed upon a man, aged forty-three years, who had acromegaly which had evidently arisen from hyperplasia of the hypophysis. There was seen hyperplasia of the tissues of the hands, feet, and face, and of many internal organs, especially of the right side of the tongue, larynx, and trachea, of the hypophysis, liver, spleen, right kidney, heart, thyroid, and pancreas—in short, splanchnomegaly. Further, there was hemiatrophy of the left side of the face, of the larynx, and of the trachea, which Harbitz does not feel competent to explain; but in the absence of a discoverable lesion of the trigeminal nerve, of any muscular lesion, and of syringomyelia, he believes that these atrophies must depend upon a lack in the *anlagen* of the organs concerned, especially since the left kidney was similarly affected. More than a coincidence is the fact that not only the hyphophysis and the thyroid were enlarged, but the pancreas and other organs of internal secretion. Harbitz recalls the effect of the hypophyseal secretion on structural growth in general, and the repression of acromegaly after removal of tumors of the organ, as evidenced by Hochenegg, Cushing, etc., as well as the linking that seems to exist between the hypophysis and thyroid on the one hand, and obesity and the failure in genital functions on the other, or again the concurrence of the two last, which Frölich united in the title of *dystrophia adiposa genitalis*. Damage to the glial portion of the hypophysis appears to cause these genital hypoplasias. Harbitz seems to have brought forward another of these interesting cases that appear to show that the glands of internal secretion are intimately associated, and that there is a "polyglandular syndrome," often caused by "pluriglandular insufficiency," but sometimes by what appears to be rather "oversufficiency" of the same structures. Some interesting certainties are arising in this field of unproved suppositions.

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